

Re-entry via Mahaim fibres as a possible basis for tachycardia

PAUL TOUBOUL, RONALD M. VEXLER,¹ AND MARIE T. CHATELAIN

From the Laboratory of Clinical Electrophysiology, Hôpital Cardiovasculaire et Pneumologique, Lyon, France

SUMMARY Paroxysmal tachycardia with widened QRS complexes was recorded in a 21-year-old man. In sinus rhythm there was no evidence of pre-excitation. His bundle studies revealed an abnormally short HV interval of 30 ms. Premature atrial stimuli produced an increased PR interval. At short coupling intervals the His bundle activity became incorporated within the QRS complex. Concurrently, a left bundle-branch block pattern appeared identical to that seen during tachycardia. Closely coupled ventricular extrastimuli initiated a tachycardia identical to the initial episode. A re-entry mechanism via anterograde Mahaim fibres and retrograde His bundle -AV node pathway is postulated.

The use of intracardiac stimulation in the study of arrhythmias has facilitated the electrophysiological identification of atrioventricular re-entry tachycardias. The fact that induced premature atrial or ventricular impulses have been shown to initiate as well as terminate such tachycardias has been held as support for the existence of these circuits (Massumi *et al.*, 1967; Durrer, 1968; Bigger and Goldreyer, 1970; Coumel *et al.*, 1970). Circus movement can revolve within the atrioventricular node itself (Goldreyer and Bigger, 1971; Denes *et al.*, 1973), or may use a combination of normal and accessory pathways of conduction. Though the latter are most often direct communications between the atria and ventricles (Kent) (Durrer *et al.*, 1967; Castillo and Castellanos, 1970; Grolleau *et al.*, 1970; Wellens *et al.*, 1971; Touboul *et al.*, 1972), atrio-His bundle accessory pathways (James) have also been invoked in the generation of re-entrant rhythms (Castellanos *et al.*, 1971; Mandel *et al.*, 1971; Coumel *et al.*, 1972b). In addition, the initiation of reciprocal excitation within an anatomical circuit composed of normal atrioventricular and accessory Mahaim pathways remains a possibility (Durrer *et al.*, 1970). Using electrophysiological data, the present report supports the existence of such a circuit underlying a junctional tachycardia.

Case report

A 21-year-old man was known to have had frequent, short episodes of tachyarrhythmia for three years before this study. In October 1974, he presented to a local emergency department complaining of an attack of 48 hours duration accompanied by dizziness, palpitations, vomiting, and abdominal pain. The electrocardiogram showed a regular tachycardia, rate 160/min. A left bundle-branch block configuration was noted (Fig. 1). After an initial failure with intravenous lanatoside C (0.8 mg), the arrhythmia was successfully and abruptly terminated by vagomimetic adenosine triphosphoric acid, 20 mg, administered intravenously. The patient was subsequently transferred to our hospital for further investigation.

Physical examination on admission was entirely unremarkable. The chest x-ray film showed a slightly enlarged cardiac silhouette. An electrocardiogram, in sinus rhythm, indicated a PR interval of 0.16 s, the QRS complex was of normal duration but increased amplitude, and there were diffuse repolarisation changes suggestive of ischemia. Vectorcardiography showed slow initial forces of ventricular depolarisation.

The patient underwent electrophysiological studies on 28 November, 1974. Informed consent had previously been obtained. Four bipolar

Dr. Vexler is a Fellow of the Medical Research Council of Canada.

catheters were introduced percutaneously, two via each femoral vein. The first was positioned across the tricuspid valve to record a His bundle electrogram. Two others were placed in the right atrium to stimulate the upper third of the interatrial septum and to record a high right atrial electrogram at the junction of the superior vena cava and lateral border, respectively. The fourth catheter was used to stimulate the right ventricle from an apical

position. Recordings were made on an 8-channel direct ink jet writing apparatus¹ at paper speeds of 100 and 200 mm/sec. A programmable modular stimulator² delivered rectangular impulses of 1.5 ms duration at an intensity equal to twice the diastolic threshold.

In sinus rhythm, at an RR interval average of

¹Elema-Schonander, Sweden.

²Tektronix, Oregon, USA.

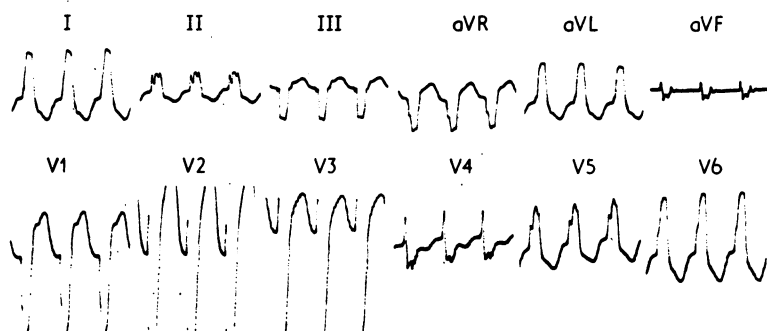


Fig. 1 Electrocardiogram during tachycardia.

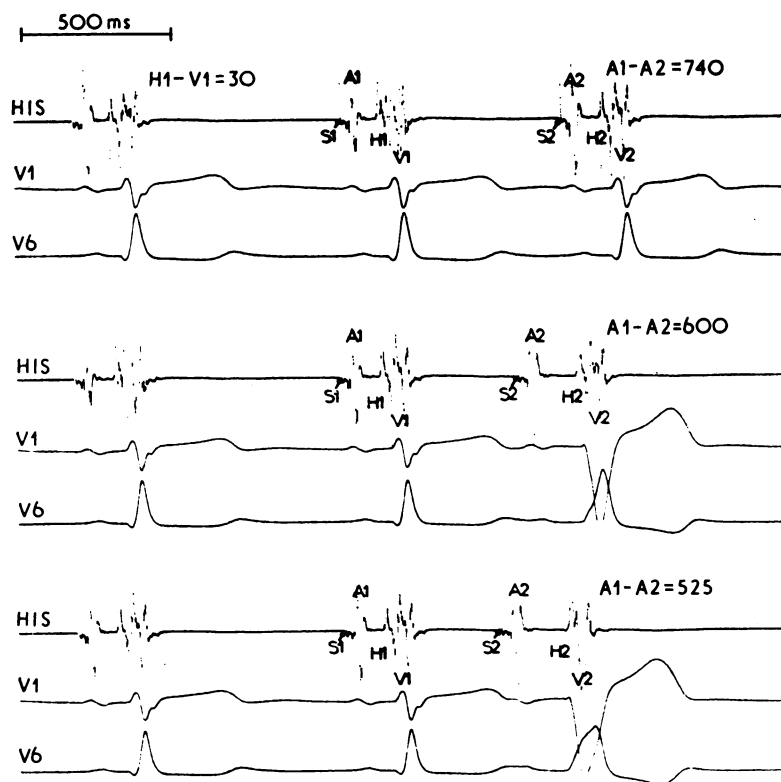


Fig. 2 Extrastimulus method. The driving cycle length is 900 ms. A1 and A2 represent the basal and premature atrial depolarisation, respectively, produced by stimuli S1 and S2. Reducing the coupling interval (S1-S2) from 740 to 525 ms is followed by an increased atrioventricular delay and the incorporation of the His activity within the ventricular complex, which itself develops a left bundle-branch block pattern. His: His bundle lead. The values given are in milliseconds.

1200 ms, the AH and HV intervals were 80 ms and 30 ms, respectively. Use of the extrastimulus method produced a gradual increase in the delay between the electrical impulse and the onset of QRS as the atrial stimuli became progressively more premature. At coupling intervals shorter than 680 ms, while the AH interval continued to increase, the HV interval actually diminished. Ultimately, the His deflection became incorporated within the ventricular electrogram. These changes were accompanied by the appearance of a left bundle-branch block pattern on the surface electrocardiogram, identical to that seen during spontaneous tachycardia (Fig. 2). Rapid atrial pacing at increasing rates gave similar results.

Right ventricular pacing was performed using programmed ventricular extrastimuli at a basic cycle length of 900 ms. Increasing prematurity to a coupling interval of 450 ms produced a ventriculo-atrial (VA) conduction time increase of only 10 ms. However, at a coupling interval of 440 ms, VA time increased abruptly in conjunction with the appearance of a retrograde His deflection. This

sequence initiated a paroxysm of tachycardia resembling the recorded spontaneous arrhythmia. A 1:1 inverted atrial activity was apparent. More premature stimuli produced similar results (Fig. 3). In every instance the QRS complex initiating the tachycardia was accompanied by a late atrial retrograde response based on an increased VH time. Subsequently, the His deflection became incorporated in the ventricular complex and was ultimately seen to appear 15 ms after the onset of the QRS complex, the HA interval being unchanged (50 ms). Concurrently the RR interval decreased from 400 to 320 ms.

These induced episodes of tachycardia either stopped spontaneously (in which case the last QRS was not followed by atrial activity), or were interrupted by a salvo of rapid ventricular stimuli. As well, rapid atrial stimulation during the tachycardia at a driving cycle of 200 ms permitted capture of the ventricles with consequent slowing due to a 2:1 atrioventricular block. The QRS complexes thus produced were identical to those during the tachycardia.

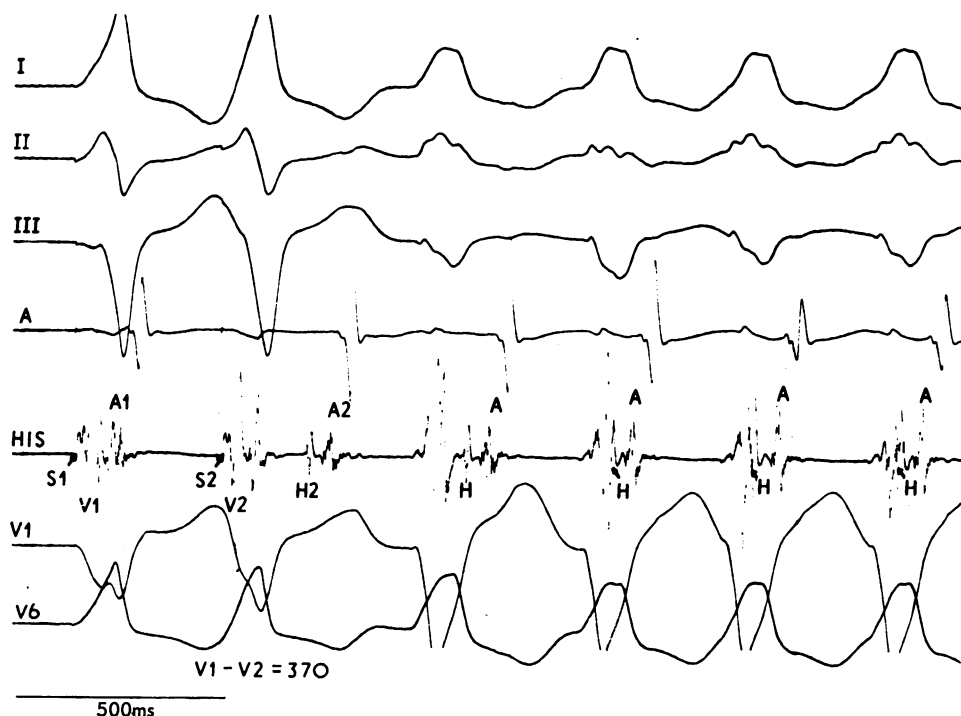


Fig. 3 Tachycardia induced by a ventricular extrastimulus. The coupling interval is 370 ms. The VA conduction time for the premature beat is prolonged and a retrograde His bundle potential can be clearly seen detached from the ventricular activity. Thereafter, as the rate of the tachycardia increases, the His deflection is gradually incorporated within the QRS.

Finally, attempted stimulation in the region of the common bundle failed to provide useful information.

During his stay in hospital the patient also underwent complete cardiac catheterisation and coronary angiography which suggested the diagnosis of a primary myocardial disease process. He has been followed for one year during which time five further episodes of tachycardia have been reported. Treatment has consisted of quinidine bisulphate orally 1 g/day which he has taken irregularly.

Discussion

The concept of a partial bypass of the atrioventricular conducting system by septal fibres originating from the distal atrioventricular node or proximal His bundle has been anatomically proven (Mahaim and Winston, 1941; Lev *et al.*, 1966, 1975). The classic electrocardiographic signs held to be associated with such a phenomenon include a delta wave accompanied by a normal or prolonged PR interval as well as the persistence of the pre-excitation pattern with junctional beats. The latter criterion was sufficient proof for Pick and Katz (1955) that such connections existed. Recent electrophysiological studies have aided in the understanding of this syndrome (Castillo and Castellanos, 1970; Coumel *et al.*, 1972a; Touboul *et al.*, 1975). The shortness of HV, the lengthening of atrioventricular conduction time with increased pacing rates, and the production of a pre-excitation pattern by His bundle stimulation are all arguments in support of a His bundle ventricle bypass. Other authors have also described conduction via Mahaim fibres which produced a His bundle deflection after the onset of QRS activity (Tonkin *et al.*, 1975). In that particular study, stimulation in the region of the His bundle was accompanied by the disappearance of the delta wave and normalisation of the QRS complex. These results were explained by the emergence of the accessory fibres at the atrioventricular nodal level, that is, proximal to the level of His bundle stimulation. As for the anomaly of ventricular depolarisation, previous reports have shown that it may increase (Wellens, 1971), diminish (Touboul *et al.*, 1975), or remain unchanged (Castillo and Castellanos, 1970) during atrial pacing.

Mahaim fibres are rarely implicated as causing re-entrant activity. Two cases have been reported in which a reciprocal rhythm used these fibres in an anterograde direction and a Kent pathway for retrograde conduction (Castillo and Castellanos, 1970; Tonkin *et al.*, 1975). As to the possibility that circular movement could exist between Mahaim fibres and the His bundle itself, one case has been

described in a child, and another more recently which were suggestive of such a phenomenon (Wellens, 1971; Tonkin *et al.*, 1975).

In the present study, a functioning accessory pathway between the atrioventricular node and right ventricular myocardium or right bundle-branch system seems likely. The absence of abnormal QRS complexes on the surface electrocardiogram during sinus rhythm can be explained by a minimal degree of pre-excitation in the basal state and is consistent with an HV interval approaching the lower limits of normal. Results obtained with premature atrial stimulation suggest that, at long coupling intervals, atrioventricular nodal delay is situated proximal to the point of origin of the anomalous tract. Thus prolongation of atrioventricular conduction time is related directly to an increased AH interval, while HV remains unchanged. But with progressively premature stimuli, an additional area of atrioventricular nodal delay seems to appear, this time distal to the origin of the Mahaim fibres. The impulse traversing this region reaches the His bundle later and later, allowing accessory pathway conduction to play an enhanced role in ventricular activation. In this way the His bundle deflection can be seen to appear after the onset of the QRS complex which, itself, takes on the configuration of aberrant ventricular depolarisation.

The initiation of tachycardia by ventricular extrastimuli can be explained by a dissociation in the refractory periods of the Mahaim and normal pathways. A sufficiently premature ventricular depolarisation finds the Mahaim pathway refractory and is conducted retrogradely solely by the His bundle and atrioventricular node. The impulse is then able to re-excite the ventricles by way of the accessory fibres and the re-entry circuit is primed. The pronounced abnormality of the QRS complexes is related to exclusive Mahaim conduction. There is a gradual acceleration of conduction within the circuit as witnessed by the decreasing RR interval and the migration of His bundle activity to an earlier position. Sympathetic stimulation (Dhingra *et al.*, 1973; Vargas *et al.*, 1975) and rate-dependent decrease in His-Purkinje refractory periods (Denes *et al.*, 1974) could account for this phenomenon. The ability to interrupt the attacks by either atrial or ventricular stimulation lends credence to the concept of re-entry within the atrioventricular junction. Additional proof that the AV node is included in the re-entry circuit is offered by the effectiveness of vagomimetic drugs in terminating the arrhythmia.

During tachycardia, the atrioventricular interval is prolonged suggesting slow conduction through the anomalous pathway. But this is also consistent

with the location of greatest conduction delay at the atrioventricular nodal insertion of the Mahaim tract. As for the short HA time, it might be caused by retrograde involvement of a concealed fast pathway within or parallel to the atrioventricular node (Narula, 1974).

Among other factors there is the possibility of a circular movement confined to the atrioventricular nodal area. Such a mechanism is unlikely, owing to the absence of significant HA interval prolongation with the initiating ventricular premature beat. Likewise a re-entry within the His-Purkinje system should be considered (Wellens *et al.*, 1972; Spurrell *et al.*, 1973; Akhtar *et al.*, 1974). Thus the premature impulse before tachycardia might find the right bundle-branch and Mahaim tract refractory and would then proceed across the ventricular septum and retrogradely along the left bundle-branch and His bundle. This sequence would occur slowly enough to allow recovery of the right conducting system, permitting ventricular re-excitation as well as retrograde invasion of the Mahaim tract. The circuit could then continue in a transseptal fashion. However, the final situation of the His potential 15 ms only after the onset of the QRS complex does not fit such a concept. In this view, micro-re-entry occurring in the right ventricle would be more plausible, but the effects of atrial pacing during tachycardia do not support this hypothesis. Anyhow, the role of vagal action in terminating the arrhythmia argues against a ventricular origin, though this proof is not definite (Waxman *et al.*, 1974). Moreover, the latter explanation does not take into account the similarity between the pre-excitation beats induced by atrial premature stimulation and those seen during tachycardia.

If our hypothesis is correct, we are dealing with a supraventricular mechanism, despite the absence of His deflection before the wide QRS complexes. This is a well-known phenomenon in the tachycardias occurring in the Wolff-Parkinson-White syndrome, where atrioventricular conduction involves the Kent bundle (Narula, 1973; Wellens, 1975). The present case is further misleading because the surface electrocardiogram showed no evidence of pre-excitation in sinus rhythm. Moreover, theoretically, one would expect that His bundle pacing during the attacks would normalise the QRS complex. Such a response had previously been assumed to prove the existence of ventricular tachycardia (Puech *et al.*, 1970).

Finally, a similar circuit operating in the reverse direction is equally conceivable. An impulse could proceed to the ventricles over the normal pathway, then continue in the reverse direction via a Mahaim tract exhibiting anterograde block. No such schema

has yet been shown as the basis for reciprocating tachycardias. Nevertheless, it may be implicated in the origin of atrial echo beats which are dependent on a critical conduction delay within the His-Purkinje system (Varghese *et al.*, 1974).

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Requests for reprints to Dr. Paul Touboul, Hôpital Cardiovasculaire et Pneumologique, BP Lyon Montchat, 69394 Lyon Cedex 3, France.